

SECTION IV



Figure 4. Occupational contact dermatitis.

DERMATOSES

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Any abnormality of the skin induced or aggravated by the work environment is termed an "occupational dermatosis." A dermatosis, therefore, can represent one or more alterations in the skin ranging from the mildest erythema and scaling to a complicated eczematous, acne-form, pigmentary, neoplastic, granulomatous, or ulcerative disorder. Commonly used terms as "industrial dermatitis" or "occupational contact dermatitis" actually designate an inflammatory process of eczematous nature; whereas descriptive titles as "cutting oil acne," "tar melanosis," "pitch cancer," "silica granuloma," "chrome ulcer," among others, are used to identify cause and effect.

Occupational diseases of the skin are common for at least two reasons: first, the skin has a large surface area available for contact exposure; and, second, the work environment contains innumerable natural and artificial materials capable of exerting chemical, mechanical, physical, biological, or photoreactive insults to the skin. Causal agents, therefore, are diverse; but chemicals are by far the most frequent offenders.

Organic and inorganic chemicals are everywhere in modern industry, on the farm, and even in the household. In acting as primary irritants or allergic sensitizers or as photosensitizers, the usual clinical effect is contact eczematous dermatitis. This type of cutaneous response characterizes about 75 to 80 per cent of the occupational dermatoses observed and about four-fifths of these are due to contact with primary irritant chemicals. See Figure 4.

NATURAL PROTECTION

Anyone who works is a potential candidate for developing an occupational dermatosis; however, many workers are not affected with these disorders. Several intrinsic factors influence the behavior of the worker's skin, the most important of these is its own natural defense. Anatomically, the skin acts as a body envelope whose connective tissue, including the elastic fibers, provides flexibility which buffers moderate blunt mechanical trauma. The outermost layer of the skin, thickest on the palms and soles, is a complex protein called "keratin." Though composed of dead cells, the keratin layer is a most important line of cutaneous defense. It protects against the rapid entrance of water and water-soluble chemicals and, similarly, prevents mass loss of water from the body. In a limited way, it offers resistance to mild acids and water; but its chemical nature does not furnish satisfactory defense against alkalis, strong acids, solvents, or prolonged immersion. These agents disrupt the chemical nature and the cohesiveness of the keratin cells, thereby, weakening their barrier effect. Protection also results from the increased thickening of the keratin layer which follows repetitive trauma or the action of ultraviolet rays.

Keratin cells are covered by a film of sweat and sebaceous gland material (sebum) mixed with the by-products of the keratinizing epidermis. All of these materials make up the acid surface film, sometimes called the "acid mantle." This film may provide some impedance to the rapid entrance of water and water-soluble chemicals; but its ready removal by water, soap, detergents, and solvents obviously limits its effectiveness in defense.

Residing below the keratin layer are the living epidermal cells which stem from the basal cells located in the lowermost area of the epidermal layer. Basal cells or germinative cells supply the epidermal cells, which in turn, become keratin cells. Located among the basal cells are the pigment-forming elements, the "melanocytes," which manufacture melanin. This material, the product of an enzyme reaction, is picked up by the epidermal cells and migrates upward with them, at the same time furnishing protection against sunlight and ultraviolet rays.

Blood vessels, nerves, sweat glands, and sebaceous glands are located within the dermis. The major defense against heat is furnished by the interaction of blood vessels, nerves, and sweat glands. Located within the upper dermis are numerous nerve receptors and effectors which delineate sensory perception and, thereby, afford protection against certain external stimuli. An antimicrobial capacity appears to be present in the skin and probably is influenced by the skin's secretions and excretions, products arising from the cellular turnover of the epidermal cells, and the inherent dryness which the skin possesses.

PRIMARY IRRITANTS

Most occupational dermatitis is caused by primary irritant chemicals. These agents will cause dermatitis by direct action on the normal skin at the site of contact if they are permitted to act in sufficient intensity or quantity for a sufficient length of time. Thus, any normal skin will almost always react to a primary irritant if the above requirements are met. Strong or absolute irritants as sulfuric acid, sodium hydroxide, or methyl bromide produce an observable effect within a few minutes, depending upon their concentration. Conversely, weak or marginal irritants as acetone, soap and water, and mineral oil may require several days before a clinically recognizable change occurs.

The precise mechanism of how primary irritants disturb the skin is far from understood, but clinical effect generally varies with the degree of injury. Strong acids react vigorously with skin to form acid albuminates. Clinically, an acid burn resembles a thermal burn. Strong alkalis combine with fats and also dissolve protein, including the cytoplasm of keratin cells. Marginal alkaline irritants produce less destruction of tissue, but subtly cause dehydration of the keratin by inducing loss of cell cohesion, cracking, and loss of continuity.

Lipid solvents remove the surface film, disturb the water-holding quality of the keratin cells, and injure the membranous structure of epidermal cells. Metallic salts of arsenic, mercury, zinc, and chromium

precipitate skin protein and if the concentration of these materials is sufficient, ulceration of the skin occurs. Otherwise, contact dermatitis results. Oxidizing and reducing agents also injure keratin and epidermal cells. The events which occur in the various layers following exposure to most of the irritant chemicals are not well understood.

ALLERGIC SENSITIZERS

It is generally estimated that 20 per cent of occupational contact dermatitis is caused by allergenic materials. Naturally, this will vary in any industrial plant, depending upon the materials being handled. Practically any chemical can act as a sensitizer, but certain ones are more conspicuous because of this capacity. Some examples are: poison ivy, poison oak, epoxy monomers and their amine hardeners, potassium dichromate, nickel, formaldehyde, tetramethylthiuram disulfide, and mercaptobenzothiozole.

Cutaneous sensitizers differ from primary irritants in their mode of action and in the effects they create within the skin. Most sensitizers do not produce demonstrable cutaneous changes on first contact or perhaps for many days or weeks. However, the sensitizer induces certain specific cellular changes in the skin so that after a period of incubation (five to seven days or more) further contact with the same or a closely related agent on the same or other parts of the body results in an acute dermatitic reaction.

The essential differences between primary irritation and allergenic sensitization, therefore, are time and a different mechanism of action. Another difference which may be noted is that an irritant usually affects a number of workers whereas a sensitizer generally affects a few. This, of course, does not apply to potent sensitizers as poison ivy or epoxy resin systems. In spite of these differences, it can be extremely difficult to distinguish between a marginal irritant and a cutaneous sensitizer because the marginal irritant also requires prolonged or repeated contact before a dermatitis appears.

PHOTOSENSITIZERS

Photosensitivity is the capacity of an organ or organism or certain chemicals and plants to be stimulated to activity by light or to react to light. Photosensitizers are divided into "phototoxic" and "photoallergic" materials. Several derivatives of coal tar, e.g., anthracene, phenanthrene, and pitch, possess this activity.

Photobiologic effects as melanosis or photodermatitis are associated with specific chlorinated hydrocarbons, particularly the chlorobenzols, the diphenyls, and the triphenyls; dyes as acridine, eosin, and rose bengal; certain essential oils as bergamot; and a number of plants as limes, wild parsnip, fennel, and "pink-rot" celery.

Phototoxicity, like primary irritation, can affect anyone; however, heavily pigmented skin is more resistant. Similarly, photoallergens, like allergic sensitizers, affect far fewer people.

DIAGNOSIS

Recognizing and diagnosing an occupational dermatosis, like any cutaneous disease, depends upon satisfying several basic criteria. These fundamental tenets deal with the morphology or appearance of the eruption, its location, the history and course of the disease, and laboratory data.

The eruption should look like a contact dermatitis or one of the other clinical types classified as an occupational dermatosis. Lesions should be located on sites of greatest exposure, notably the backs of the hands, the volar surfaces of the wrists, the forearms, and the cubital areas—particularly when dusts and liquids are the contactants. Involvement of the face, eyelids, ears, and “V” of the neck usually indicates exposure to fumes, vapors, or mists. Excessively dusty exposures can affect large areas of the skin by direct contact or by manual transmission.

When the disease is suspected of being occupational, the history should reveal that the rash occurred after beginning work and that periods of remission and recurrence correlate with work exposure. A thorough history reveals the substances being handled at work and at home, including medications applied or ingested; the manner in which contact has been made; cleansing habits with soaps or solvents; and the use of protective equipment, including barrier creams, gloves, etc.

Most workers with an active occupational dermatosis relate that the eruption diminishes, but rarely disappears completely, over the weekend. Additionally, the eruption almost always worsens upon return to work after the weekend. When the eruption fails to clear after six to eight weeks of sick leave or after complete withdrawal from the suspected contact agent through a job change, other reasons for the dermatitis should be explored.

Actually, the prolonged or recurrent eruption may represent an underlying disorder as atopic dermatitis, psoriasis, nonoccupational contact dermatitis, nummular eczema, among other nonoccupational diseases. Certain laboratory procedures may be indicated and may further define the disease process. These may include skin scrapings and culture for detecting the presence of a superficial fungus, anaerobic or aerobic cultures for bacteria, histopathologic diagnosis following skin biopsy, diagnostic patch tests or photopatch tests to detect an allergen. Additional analytical tests for porphyrins in urine and for metals in urine or blood, skin or hair may also be indicated.

CLINICAL CLASSIFICATION

Occupational dermatoses have considerable morphologic variety. Their appearance and pattern rarely indicate the provoking substance, but may provide a clue as to a class of materials being encountered. Diagnosis depends upon appearance and location, but mostly upon history. Supervening infections or undesirable therapeutic effects make the diagnosis more difficult.

Despite the numerous clinical reactions the skin can display, the

following groupings comprise the majority of the occupational dermatoses:

(1) Acute contact eczematous dermatitis characterized by erythema, edema, papules, vesicles or bullae, crust, scale, and finally, desquamation. These are the signs of an inflammatory eczematous dermatitis caused by contact with a primary irritant or a sensitizer or a photosensitizer.

(2) Chronic eczematous dermatitis characterized by erythema, lichenification, scaling, dryness, and fissuring resulting from contact with substances which dehydrate the skin as alkali, liquids and dusts, solvents, soaps and detergents.

(3) Folliculitis and acneform dermatoses including chloracne characterized by plugged sebaceous follicles and nodular and suppurative lesions. Chloracne also shows multiple cystic lesions which contain straw-colored material. These dermatoses are caused by contact with insoluble oils, greases, tars, waxes, and certain chlorinated hydrocarbons as the chloronaphthalenes.

(4) Neoplastic (benign and malignant) types as keratoses, papillomata, epitheliomas, and carcinomas of the exposed areas. These usually are caused by certain petroleum products, coal tar and certain derivatives, sunlight, and ionizing radiation.

(5) Pigmentary disturbances characterized by an increase or decrease of pigment in the epidermis. Increased pigmentation can result from contact with coal tar compounds, certain petroleum oils, vegetables, fruits, sunlight, and trauma. Decreased or absent pigmentation may result from burns; forceful trauma; chronic dermatitis; monobenzyl ether of hydroquinone; and certain phenolics as tertiary butyl catechol, tertiary amyl phenol, and tertiary butyl phenol.

(6) Granulomatous dermatoses characterized by chronic indolent focal inflammations which tend to heal with scar. These lesions can result from bacterial, viral, fungal or inanimate agents as asbestos, beryllium, and silica.

(7) Ulcerative lesions characterized by a loss of tissue on a cutaneous or mucous membrane surface leading to necrosis. Ulcerations can be caused by arsenic trioxide, calcium compounds, cement and concrete, chromic acid, burns and trauma. They may also result from purposeful or unconscious manipulation.

(8) Miscellaneous lesions. Some occupational dermatoses, because of their unusual nature, do not fit into the above classifications. Among such miscellaneous lesions are:

- (a) alopecia induced by chloroprene;
- (b) acro-osteolysis, with or without Raynaud's;
- (c) sclerodermoid changes believed due to vinyl chloride polymerization;
- (d) discolorations of the hair, skin, and nails due to various chemicals;
- (e) porphyria cutanea tarda caused by a certain chlorinated hydrocarbon intermediate.

OCCUPATIONS AND AGENTS

The following is a list of occupations each accompanied by certain agents frequently associated with that occupation and capable of producing a dermatosis. Additional agents for the occupations listed as well as additional occupations will be found in other sections, principally the one on chemical hazards.

Abrasive Wheel Makers

carborundum
emery
resin glues

insecticides
medicaments
parasites
pesticides
viruses

Agricultural Workers

See Farmers

Artists (Painters)

acrylics
epoxies
paint removers
pigments
plasticizers
solvents

Aircraft Workers

adhesives (resins)
alkalis
bichromates
chromates
chromic acid
cutting fluids
cyanides
epoxy resins
flame retardants
glass fibers
hydraulic fluids
hydrofluoric acid
lubricants
nitric acid
oils
paints
plastics
rubber
solvents
thinners
ultraviolet light
vibrating tools
X-rays

Artists (Sculptors)

dusts
plaster of Paris
pneumatic tools
polishes

Athletes

adhesives
antibiotics
bacteria
lime
medications
protective gear
soaps

Animal Handlers

antibiotics
bacteria
cleaners & detergents
deodorants
feeds
fungi
germicides

Automobile Workers (Assembly)

adhesives
asbestos
antifreeze
brake fluids
brake linings
flame retardants
gasoline
hydraulic fluids
oils
rubber
solvents

Automobile Workers (Body)

abrasives
 adhesives
 alkalis
 lead
 paints
 rubber compounds
 solder
 solvents

Automobile Workers (Mechanic)

acids
 adhesives
 alkalis
 antifreeze
 brake fluids
 brake linings
 cleansers
 epoxy resins
 gasoline
 hydraulic fluids
 lubricants
 rubber
 solvents
 thinners

Bakers

benzoyl peroxide
 cinnamon
 dough
 dusts
 flavors (oils)
 flour
 fungi
 heat
 moisture
 spices
 sugar

Barbers

ammonium thioglycolate
 antiseptics
 bacteria
 cosmetics
 depilatories
 detergents
 dyes
 fungi

hair conditioners
 hair sprays
 hair straighteners
 hair tonics
 perfumes
 shampoos
 shaving creams
 ultraviolet
 vibrating machines
 wave solutions

Bartenders

citrus fruits
 detergents
 disinfectants
 flavors
 moisture
 soaps

Bath Attendants

deodorants
 fungi
 liniments
 lotions
 oils
 soaps
 ultraviolet

Battery Makers

alkali
 cobalt
 epoxy sealer
 fiber glass plates
 mercury
 moisture
 nickel
 pitch
 plastics
 solvents
 sulfuric acid
 zinc chloride

Bleachers

borax
 chlorine compounds
 hydrochloric acid
 hydrogen peroxide
 oxalic acid

Bleachers (cont'd)

per-salts
potassium hydroxide
sodium hydroxide
solvents

Bookbinders

formalin
glues (natural)
glues (resin)
inks
shellac
solvents

Brick Masons

cement
chromates
cold
epoxy resins
lime
moisture
sunlight

Briquette Makers

coal tar pitch

Bronzers

acetone
ammonia
ammonium sulfide
amyl acetate
antimony sulfide
arsenic
arsine
benzine
benzol
cyanides
heat
hydrochloric acid
lacquers
mercury
methyl alcohol
petroleum hydrocarbons
phosphorus
resins
sodium hydroxide
sulfur dioxide
turpentine
varnishes

Broom Makers

bacteria
bleaches
dust, vegetable
dyes
fungi
glues - natural & resin
parasites
pitch
plastics
rubber
shellac
solvents
tar
varnish
woods

Brush Makers

See Broom Makers

Butchers

antibiotics
bacteria
brine
cold
detergents
enzymes
fungi
moisture
parasites

Button Makers

bacteria
dusts - animal, vegetable,
mineral
dyes
hydrogen peroxide
plastics

Cabinet Makers

bleaches
glues - resin & casein
insulation agents
oils
polishes
rosin
shellac
solvents

Cabinet Makers (cont'd)

stains
woods

Cable Splicers

chlorinated diphenyls
chlorinated naphthalenes
dyes
epoxy resins
solvents

Cable Workers

See Cable Splicers

Candle Makers

ammonium salts
borax
boric acid
chlorine
chromates
hydrochloric acid
potassium nitrate
sodium hydroxide
stearic acid
waxes

Candy Makers

chocolate
citric acid
dyes - food
essential oils (flavors)
fruits
pineapple juice
spices
sugar
tartaric acid

Canners

bacteria
citrus oil
dyes
enzymes
fruit acids & sugars
fungi
moisture
parasites
resins
salt
vegetable juices

Carpenters

See Cabinet Makers

Carpet Makers

alizarine dye
aniline dyes
anthrax bacillus
bleaches
chlorine
fungicides
glues
insecticides
jute
loom oils
solvents

Carroters - Felt Hat

acids
anthrax bacillus
quinones

Case Hardeners

heat
oils (quench)
sodium carbonate
sodium cyanide
sodium dichromate
sodium nitrite

Cellulose Workers

acids
alkalis
bleaches
carbon disulfide
finishing oils

Cement Workers

cement
chromates
cobalt
epoxy resins
lime
moisture
pitch
resins

Chemical Workers

See Section VII

Chrome Platers

chromium compounds
degreasers (solvents)
metal cleaners (alkali)
sulfuric acid

Clerks

adhesives
carbon paper
copy paper
duplicating fluids
duplicating materials
indelible pencils
ink removers
inks
rubber
solvents
type cleaner
typewriter ribbons

Cloth Preparers

acids
alkalis
amino resins
detergents, synthetic
dyes
flame retardants
formaldehyde
fungicides
moisture
potassium salts
soaps
sodium metasilicate
sodium salts
sodium silicate

Coal Tar Workers

anthracene oil
benzol
coal tar
creosote
cresol
naphtha
pitch
solvents
sunlight

Compositors

alkalis

inks
solvents

Construction Workers

adhesives, resin
cement
concrete
cold
creosote
gasoline
glass fiber
oils
paints
pitch
poisonous plants
sealers
solvents
sunlight
ultraviolet light
wood preservatives
woods

Cooks

fruit acids
heat
moisture
monilia
spices
sugars
vegetable juices

Cotton Sizers

acids
aluminum salts
arsenic salts
calcium salts
dicyandiamide formaldehyde
fungicides
magnesium salts
melamine formaldehyde
sodium hydroxide
starch
urea formaldehyde
zinc chloride

Dairy Workers

antibiotics
bacteria

Dairy Workers (cont'd)

deodorants
detergents
fungi
mites
viruses

Degreasers

alkalis
chlorinated hydrocarbon
solvents
petroleum solvents
ultrasonic devices

Dentists

anesthetics, local
antibiotics
bacteria
disinfectants
eugenol
ionizing radiation
mercury & metallic amalgams
oil of clove
resins
soaps
waxes

Dishwashers

bacteria
detergents, synthetic
grease
moisture
monilia
soaps
water softeners

Disinfectant Makers

carbolic acid
chloride of lime
chlorinated phenols
chlorine
cresol
formaldehyde
iodine
mercurials
quarternary ammonium
compounds
surfactants
zinc chloride

Dock Workers

bacteria
castor bean pomace
chemicals
cold
fumigants
fungi
grains
heat
insecticides
insects
irritating cargoes
mites
moisture
petroleum
sunlight
tar

Druggists

acids
alkalis
antibiotics
bleaching powder
detergents, synthetic
drugs
iodoform
soaps
sugar

Dry Cleaners

acetic acid
ammonia
amyl acetate
benzine
carbon tetrachloride
dusts
methanol
nitrobenzene
perchloroethylene
sizing chemicals
Stoddard solvent
trichloroethylene
turpentine
waterproofing chemicals

Dye Makers

acids
alkalis

Dye Makers (cont'd)

antimony compounds
benzine
calcium salts
coal tar products
cresol
dextrins
dye intermediates
ferrocyanides
formaldehyde
gums
hydroquinone
lead salts
potassium chlorate

Dyers

acids
alkalis
bleaches
detergents, synthetic
dyes
mercurial salts
moisture
solvents
zinc chloride

Electric Apparatus Makers

acids
asbestos
chlorinated diphenyls
chlorinated naphthalenes
enamels
epoxy resins
ionizing radiation
phenolic resins
pitch
rubber
solder fluxes
solvents
synthetic waxes
varnishes

Electricians

chlorinated diphenyls
chlorinated naphthalenes
electricity
epoxy resins

solder fluxes
solvents
waxes, synthetic

Electroplaters

acids
alkalis
benzine
chromic acid
heat
lime
moisture
nickel
potassium cyanide
soaps
waxes, chlorinated
zinc chloride
zinc cyanide

Embalmers

bacteria
formaldehyde
fungi
ionizing radiation
mercury
oil of cinnamon
oil of clove
phenol
thymol
zinc chloride

Enamelers

acids
alkalis
antimony
arsenic
chromium
cobalt
nickel

Engravers

acids
alkalis
chromic acid
ferric chloride
potassium cyanide
solvents
tropical woods

Etchers

acids
alkalis

Explosive Workers

ammonium salts
mercury compounds
nitroglycerin
PETN
picric acid
tetryl
TNT

Farmers

antibiotics
bacteria
cold
detergents, synthetic
disinfectants
feeds
fertilizers
fruits
fungi
fungicides
heat
lubricants
oils
paints
parasites
pesticides
poison ivy, oak, sumac
ragweed
solvents
sunlight
tar
vegetables
wood preservatives

Felt Hat Makers

acids
bacteria
dyes
Glauber's salt
hydrogen peroxide
mercuric nitrate (if used)
sodium carbonate

Fertilizer Makers

acids
ammonium compounds
calcium cyanamide
castor bean pomace
fluorides
lime
manure
nitrates
pesticides
phosphates
potassium salts

Fish Dressers

bacteria
brine
cold
moisture
redfeed
sunlight
trauma

Florists

bacteria
bulbs
fertilizers
fungi
herbicides
parasites
poisonous plants

Flour Mill Workers

chemical bleaches
dusts
parasites
pesticides

Food Preservers

bleaches
brine
ionizing radiation
moisture
monilia
resins
spices
sugar
vinegar
waxes

Foundry Workers

acids
heat
lime
resin binder systems
solvents
ultraviolet light

Fur Processors

acids
alkalis
alum
bacteria
bleaches
chromates
dyes
formaldehyde
fungi
lime
oils
salt

Furnace Workers

heat
ultraviolet light

Furniture Polishers

acids
alkalis
benzine
essential oils in polish
methyl alcohol
naphtha
pyridine
rosin
soaps
solvents
stains
turpentine
waxes

Galvanizers

acids
alkalis
zinc chloride

Garage Workers

air guns (grease)

antifreeze
detergents, synthetic
epoxy resins
gasoline
gasoline additives
glass fiber
greases
moisture
oils
paint removers
paints
solvents

Gardeners

bacteria
fertilizers
fungi
fungicides
herbicides
insecticides
insects
plants
poison ivy
poison oak
sunlight

Glass Workers

arsenic
borax
boric acid
glass fiber
glass wool
heat
hyrdofluoric acid
lead compounds
lime
metallic oxides
petroleum oils
resins
soda ash
ultraviolet light

Hairdressers

See Barbers

Highway Workers

See Road Workers

Histology Technicians

alcohol
 aniline
 benzol
 epoxy resins
 formaldehyde
 mercury bichloride
 osmium tetroxide
 potassium dichromate
 stains
 toluene
 waxes
 xylene

Ink Makers

anti-skinning agents
 (antioxidants)
 chrome pigments
 cobalt compounds (driers)
 detergents, synthetic
 dyes
 mercurial pigments
 resins
 soaps
 solvents
 turpentine
 varnishes

Insecticide Makers

aldrin
 allethrin
 arsenic trioxide
 calcium arsenate
 chlordane
 DDT
 dieldrin
 lindane
 malathion
 methoxychlor
 parathion
 piperonyl compounds
 pyrethrin
 strobane
 See also Pesticides section

Janitors

bacteria
 detergents, synthetic

disinfectants
 house plants
 polishes (essential oils)
 soaps
 solvents
 waxes

Jewelers

acids
 adhesives, resin
 chromium
 cyanides
 mercury
 mercury solvents
 nickel
 rouge
 solder flux

Laboratory Workers, Chemical

acids
 alkalis
 chromates
 detergents, synthetic
 moisture
 organic chemicals
 soaps
 solvents

Laundry Workers

alkalis
 bactericides
 bleaches
 chemical dusts
 detergents, synthetic
 enzymes
 fiber glass
 fungicides
 heat
 moisture
 optical brighteners
 soaps

Linoleum Makers

asphalt
 dyes
 oils
 pigments
 resins
 solvents

Longshoremen

See Dock Workers

Machinists

antioxidants
aqueous cutting fluids,
synthetic
chlorinated cutting oils
chromates
germicides
greases
insoluble cutting oils
lubricants
rust inhibitors
soluble cutting fluids
solvents

Masons

See Brick Masons

Match Factory Workers

ammonium phosphate
chromates
dextrins
dyes
formaldehyde
glues
gums
phosphorus sesquisulfide
potassium chlorate
red phosphorus
waxes

Meat Packers

See Butchers

Mechanics

See Aircraft Workers,
Automobile Workers, &
Garage Workers

Mercerizers

acids
alkalis
heat
moisture

Metal Polishers

abrasives

acids
alkalis
ammonia
naphtha
pine oil
potassium cyanide
soaps
soluble oils
solvents
triethanolamine
waxes
wood (pine)

Mirror Makers

acids
ammonia
cyanides
formaldehyde
lacquers
silver nitrate
solvents
tartaric acid
varnishes

Mordanters

acids
alkalis
aluminum salts
antimony compounds
arsenates
chromates
copper salts
iron salts
lead salts
phosphates
silicates
tin salts
zinc chloride

Nickel Platers

acids
alkalis
degreasers
detergents, synthetic
heat
moisture
nickel sulfate
zinc chloride

Nitroglycerin Makers

ethylene glycol dinitrate
 nitric acid
 nitroglycerin
 sodium carbonate
 sulfuric acid

Nurses

anesthetics, local
 antibiotics
 antiseptics
 bacteria
 detergents, synthetic
 disinfectants
 drugs
 ethylene oxide
 fungi
 ionizing radiation
 moisture
 rubber gloves
 soaps
 tranquilizers
 viruses

Oil Field Workers

acids
 alkalis
 brine
 crude petroleum
 explosives
 ionizing radiation
 lubricating oils
 sunlight

Optical Workers

alkalis
 grinding fluids
 oils
 turpentine

Packinghouse Workers

antibiotics
 bacteria
 bone fragments
 brine
 cold
 detergents, synthetic
 enzymes

fungi
 parasites
 spices

Paint Makers

anti-mildew agents
 chromates
 coal tar distillates
 driers
 fish oils
 latex
 oil, vegetable
 petroleum solvents
 pigments
 plasticizers
 resins
 thinners
 turpentine
 zinc chloride

Painters

acetone
 acids
 alkalis
 benzine
 chlorinated hydrocarbons
 chromates
 driers
 paint strippers
 paints, oil base
 paints, resin
 pigments
 solvents
 thinners
 turpentine

Paper Box Makers

anti-flame agents
 dyes
 glues, natural & resin
 mildew proofers
 waxes

Paraffin Workers

paraffin
 paraffin distillates
 solvents

Pencil Makers

aniline dyes
chromium pigments
glues
gums
lacquer
lacquer thinners
methyl violet
pyridine
red cedar wood
resins
solvents
waxes

Petroleum Refinery Workers

acids
alkalis
aluminum chloride
gas oil
gasoline
hydrofluoric acid
kerosene
paraffin
paraffin distillates
petroleum
petroleum solvents
tar
waxes

Photoengravers

ammonium bichromate
etching acids
inks
photo developers
solvents
ultraviolet light

Photographers

acids
alkalis
chromates
hydroquinone
methyl para-aminophenol
sulfate
para-aminophenol
paraformaldehyde
paraphenylenediamines
pyrogalllic acid

sodium hypochlorite
sodium sulfide
turpentine

Physicians

adhesives
anesthetics, local
antibiotics
antiseptics
bacteria
detergents, synthetic
drugs
fungi
ionizing radiation
soaps
tranquilizers
viruses

Pipeline Layers

burns
fluxes, welding
ionizing radiation
parasites
poisonous plants
solvents
sunlight
tar
ultraviolet, welding

Pitch Workers

heat
pitch
solvents
sunlight
tar

Plasterers

lime
moisture

Plastics and Resin Makers

See Section VII

Plumbers

adhesives
caulking compound
cold

Plumbers (cont'd)

fluxes, solder
hydrochloric acid
parasites
solvents
tar
zinc chloride

Printers

alkalis
aniline
chromates
glues
gums
inks
roller wash
solvents

Railroad Shop Workers

alkalis
antiseptics
chlorinated hydrocarbons
chromate (antioxidants)
cutting fluids
detergents, synthetic
diesel fuel oil
greases
insecticides
lacquers
lubes
Magnaflux (fluorescein)
paint
paint strippers
paint thinners
solvents
ultraviolet light

Railroad Track Workers

cold
creosote
fungicides
heat
herbicides
pitch
poisonous plants
sunlight
tar

Rayon Workers

acetic anhydride
acids
alkalis
ammonium sulfide
bleaches
calcium bisulfite
carbon disulfide
coning oils
sodium cyanide
sodium sulfide
sodium sulfite
solvents

Refrigeration Workers

ammonia
brine
chromates
cold
dry ice
ethyl bromide
ethyl chloride
glass fiber
methyl chloride
sulfur dioxide

Road Workers

asphalt
cement
cold
concrete
epoxy resins
herbicides
paint
parasites
pitch
poisonous plants
sunlight
tar

Rocket Fuel Handlers

aniline
boron hydrides
chlorine trifluoride
dimethylhydrazine
ethyl oxide
fuming nitric acid
gasoline

Rocket Fuel Handlers (cont'd)

hydrazine
hydrogen fluoride
hydrogen peroxide
kerosene
liquid oxygen

Rope Makers

alkalis
bleaches
dusts
dyes
oils
pitch
soaps
tar

Rubber Workers

accelerators
acids
activators
adhesive removers
alkalis
antimony
antioxidants
benzol
chloroprene dimers
chromium pigments
formaldehyde
heat
oils
plasticizers
resins
retarders
soaps
solvents
tar
turpentine
zinc chloride

Shipyard Workers

burns (welding)
chlorinated diphenyls
chlorinated naphthalenes
chromates
cold
fungicides

glass fiber
paint removers
paint thinners
paints
resins
solvents
tar
ultraviolet light
wood preservatives

Shoemakers (Manufacturers)

adhesives
ammonia
amyl acetate
amyl alcohol
aniline dyes
benzine
benzol
fungicides
hexane
naphtha
resins
rubbers
shoe polishes
tanning agents
waxes

Slaughterhouse Workers

See Packinghouse Workers

Soap Makers

alkalis
bacteriostats
detergents, synthetic
oils, vegetable
perfumes

Solderers

acids
cyanides
fluxes
heat
hydrazine salts
rosin
zinc chloride

Stevedores

See Dock Workers

Stockyard Workers

bacteria
fungi
insecticides
parasites

Stone Workers

cement
cold
dusts
heat
lime
vibrating tools

Sugar Refiners

acids
burlap
fungi
heat
jute
lime
sugar

Tannery Workers

acetic acid
alum
ammonium chloride
arsenic salts
bacteria
benzol
brine
calcium hydrosulfide
chromium compounds
dimethylamine
dyes, mineral
dyes, vegetable
formaldehyde
lime
oils
pancreatic extract
sodium hydroxide
sodium sulfide
solvents
sulfuric acid
tannin

Tar Workers

heat

pitch
solvents
sunlight
tar

Taxidermists

anthrax bacillus
arsenic salts
bacteria
calcined alum
fungi
mercuric chloride
parasites
solvents
tannin
zinc chloride

Temperers

oils
sodium carbonate
sodium cyanide
sodium dichromate
sodium nitrite

Tinners

paint
pitch
sunlight
zinc chloride

Typists

See Clerks

Undertakers

See Embalmers

Upholsterers

bacteria
flame retardants
fungi
glues
lacquer
lacquer solvents
methyl alcohol
parasites

Veterinarians

anesthetics, local

Veterinarians (cont'd)

antibiotics
bacteria
deodorants
drugs
fungi
mercuric chloride
parasites
pesticides
soaps & detergents
viruses

Watchmakers

acids
chromates
metal polishes
nickel
potassium cyanide
rouge
solvents

Waterproofers

aluminum sulfate
melamine formaldehyde resins
oils
paraffin
pitch
resin paints
rubber
solvents
tar
waxes

Welders

fluxes
heat
ultraviolet light

Wire Drawers

alkalis
drawing oils
lime
soaps
sulfuric acid

Wood Preservers

chlorophenols
chromates
copper compounds
creosote
cresols
mercuric chloride
phenylmercuric compounds
resins
tar
zinc chloride
zinc sulfate

Wood Workers

acid bleaches
amino resin glues
epoxy glues
fillers
formaldehyde
lacquers
oil stains
paints
phenolic resin glues
rosin
solvents
varnishes
woods

See also Cabinet Makers

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SECTION V

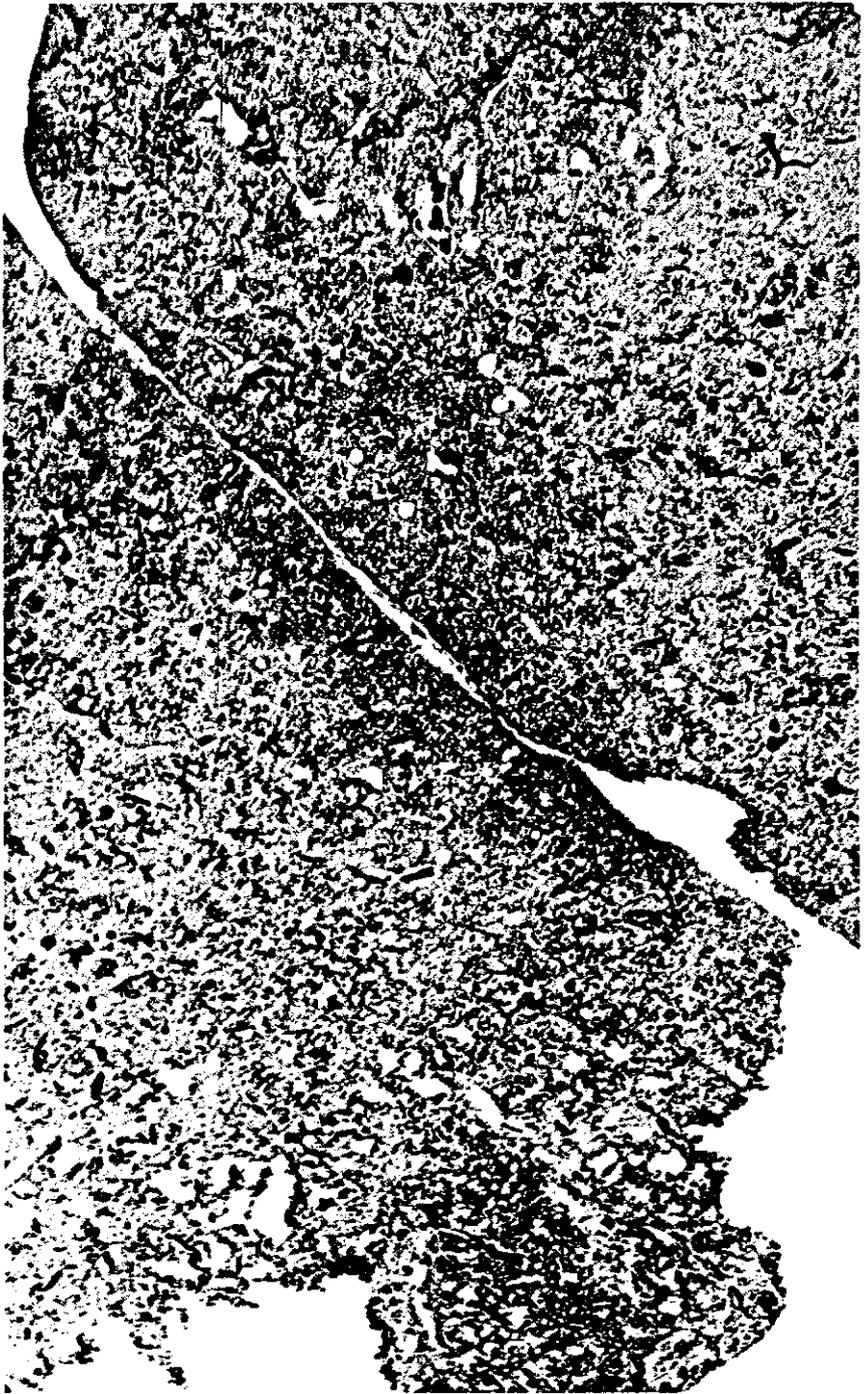


Figure 5. Coal worker's pneumoconiosis — Gough section.

DISEASES OF THE AIRWAYS AND LUNGS

W. Keith C. Morgan, M.D., and N. LeRoy Lapp, M.D.

AIRWAYS MECHANISMS AND RESPONSES

Gas exchange takes place in the acini of the lung parenchyma; that is, in those portions of the lung from the first order of respiratory bronchioles down to the alveoli. These respiratory bronchioles originate from the terminal bronchioles which are the smallest airways not concerned with gas exchange. Before inspired air can participate in gas exchange it must travel through a series of conducting tubes (the bronchial tree) until it finally reaches the first order respiratory bronchioles. The conducting system of airways does not participate in gas exchange and is, therefore, often known as the dead space. Inhaled particles may be deposited either in the lung parenchyma (the respiratory bronchioles, atrial sacs, and alveoli) or in the dead space. Some of the inhaled particulates are breathed in and out of the respiratory tract without deposition.

The site of deposition of an individual particle is governed by three factors: first, the aerodynamic properties of the particle, viz. the size, shape, speed, and density; secondly, the circumference and shape of the airway; and thirdly, the breathing pattern. Particles between 0.5 and 5 microns in diameter tend to be deposited in the alveoli and respiratory bronchioles and may, under certain conditions, cause a group of diseases known as the pneumoconioses to develop. Larger particles are in the main deposited in the conducting system of the lungs. The effect of an inhaled dust is, therefore, dependent partly on its site of deposition and partly on its toxic and antigenic properties. See Figure 5.

AIRWAYS RESISTANCE

Before considering the various occupational insults that may be inflicted upon the conducting system of the lung, certain basic anatomical and physiological considerations should be borne in mind. The resistance to air flow in the human airways can be partitioned into central and peripheral components (1). The central component comprises the resistance that is located in the upper airways, trachea, and main and segmental bronchi down to the airways that have diameters 2 mm or greater. The distal component is located in those airways whose diameter is less than 2 mm, including the gas exchanging units of the lung. Macklem and Mead have demonstrated that, of the total airways resistance, no less than 80 to 90 percent is located in the larger airways and only around 10 percent resides in the smaller airways (1). Thus, changes in the resistance to flow in the smaller airways have little influence on total airways resistance. Similarly, those indices of ventilatory capacity that are derived from the forced expiratory volume maneuver are little affected by an increase in the resistance of the small airways because these

indices for the main part reflect changes in the larger airways during dynamic compression.

Although some of the smaller air passages of the respiratory conducting system, e.g., the terminal bronchioles, are included in the peripheral airways, the remainder of the conducting system is comprised of the central airways. The respiratory symptoms and respiratory impairment that are associated with the deposition of particles in the larger airways are both more obvious and more easily demonstrated than are those associated with particulate deposition in the smaller airways (2).

RESPONSES TO DUST DEPOSITION

The deposition of inhaled dust in the central airways of the lung may induce one or more of the following four basic responses:

1. Immunologically induced airways constriction. This includes both Type I and Type III Reactions (3) and is best termed occupational asthma.
2. Pharmacologically induced airways constriction.
3. Acute irritation and reflex broncho-constriction.
4. Non-specific response to dust, viz. chronic bronchitis. This type of response is not related to the toxic properties of the dust or to its propensity to generate an immunologic reaction. Each of these responses are dealt with in turn.

IMMUNOLOGICALLY INDUCED (Asthma)

Bronchial constriction or occupational asthma may be induced by either a Type I or Type III immunological reaction (3). Type I reactions are immediate and are mediated by a specific immunoglobulin IgE. The concentration of IgE in the blood may be increased in subjects with extrinsic asthma. When a susceptible subject, viz. an atopic individual, is exposed to an antigen, there is an increase in the IgE specific to that antigen. The specific IgE binds to the mast cells present in the bronchial wall and as a result histamine and a slow reacting substance (SRS-A) are liberated. The Type III responses, which are related to the Arthus phenomenon and are associated with the presence of precipitins in the blood, occur several hours after the challenge. They are due to immunoglobulin, IgG.

Individuals with occupational asthma complain of wheeziness and shortness of breath. Initially, these symptoms occur only while the individual is at work, but later they may persist at home and on weekends. Workers who are atopic are more prone to develop occupational asthma and may do so with a relatively short exposure. Nevertheless, normal individuals may be affected although their symptoms often do not appear for several years; that is, until they have become sensitized. *Rhinitis* and *conjunctivitis* are common accompaniments of occupational asthma.

The diagnosis is made from the occupational and medical history and, if necessary, by appropriate challenge tests. When the subject is

exposed to the appropriate antigen, a decline in ventilatory capacity is usually induced. If it is a Type I reaction, the reduction in forced expiratory volume in 1 second (FEV_1) and in forced vital capacity (FVC) is usually evident within 10 to 15 minutes. If it is a Type III response, the decline is often delayed for 3 to 4 hours. Skin testing for immediate flare and wheal response is useful; however, cutaneous and bronchial responses do not necessarily correlate well with each other.

Potential exposure resulting in occupational asthma is commonly found in the following occupations: (4-17)

Grain and cereal workers, including bakers.

Woodworkers including carpenters, joiners, and sawmill operators. Western red cedar, mahogany, oak, and iroko have all been incriminated as potent sensitizing agents.

Printing. In this case gum arabic is responsible for the sensitization.

Manufacturers of detergent enzymes. These enzymes are manufactured from the products produced by fermentation of *Bacillus subtilis*. The enzyme responsible is also known as alcalase.

Soldering. This is usually due to the flux which contains amino-ethylethanolamine.

Isocyanate workers. Isocyanates are used to manufacture polyurethane foams. Two compounds have been incriminated as causes of occupational asthma. These are toluene diisocyanate (TDI) and diisocyanatodiphenyl methane (MDI).

Electroplaters, photographers, and persons exposed to platinum.

Less commonly, asthma may be associated with occupational exposures to the following: nickel, chromium, the Mexican bean weevil, locusts, silkworms, coffee, castor beans, and tungsten carbide.

PHARMACOLOGICALLY INDUCED (Byssinosis)

Airways constriction may be induced by the deposition of certain dusts in the airways in the absence of an immunological reaction (18). Thus, when certain dusts settle on the bronchial walls, the liberation of naturally occurring broncho-constrictors such as histamine and possibly serotonin may take place. Since this does not involve any immunological mechanism, the liberation of such substances can be said to be a pharmacological response to an extrinsic agent.

Although there is still some doubt, the broncho-constriction seen in byssinosis may be of this type. There is good evidence to suggest that the cotton bract contains an agent which, when it comes into contact with the bronchial mucosa, leads to the liberation of excess histamine. Byssinosis is seen in cotton, hemp, and flax workers and a similar condition possibly occurs in workers who are exposed to sisal.

The usual history of byssinosis is that the worker develops chest tightness and wheezing on return to the mill on a Monday morning. In the early course of the disease the symptoms disappear by Tuesday or Wednesday. However, with continued exposure, the tightness and shortness of breath begin to persist for longer periods until it is present all

the time. In established long standing byssinosis, the worker ends up continually short of breath and with over-distended lungs. The diagnosis is established by measuring the patient's ventilatory capacity before he starts work on Monday and again after he has finished.

IRRITANTS

If the bronchi are insulted by irritant gases or fumes, they constrict in a reflex fashion (19). Such constriction is usually accompanied by coughing, and both the coughing and bronchial constriction are mediated through vagal reflexes. Irritant gases such as chlorine, ammonia, ozone, sulfur dioxide, and the oxides of nitrogen may all produce an acute *tracheitis* and *bronchitis* which are associated with reflex broncho-constriction and coughing. With large and prolonged exposures, the lung parenchyma may also be affected. The solubility of the inhaled gas will determine whether there is a predominant proximal or distal involvement of the airways.

Aside from the above gases to which workers may be exposed as a result of industrial mishaps, certain occupational groups may be routinely exposed to other noxious fumes and aerosols in their working environment. These include: beryllium, boron hydrides (volatile), cadmium, chromium compounds (hexavalent), hydrofluoric acid, zinc chloride, manganese, mercury, osmium, and vanadium pentoxide.

DUST

Prolonged exposure to dust may lead to *industrial bronchitis* (20). The heavier the dust exposure, the more likely is the development of industrial bronchitis. The characteristic features of this condition are cough and sputum in the absence of localized destructive disease of the lungs.

The symptoms of industrial bronchitis differ in no way from those seen in chronic bronchitis due to cigarette smoking. Both conditions are characterized by production of excess mucus. The mucus is secreted by the goblet cells and more particularly the mucus glands of the bronchial tree. Airways obstruction is seen less often in industrial bronchitis than it is in the naturally occurring form of chronic bronchitis due to cigarette smoking.

Coal miners and steel workers are particularly prone to this form of airways disease. The symptoms tend to regress when dust exposure is reduced.

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EXTRINSIC ALLERGIC ALVEOLITIDES

The inhalation of organic dusts may lead to two distinct pulmonary responses. First, and more common, is that mediated by reaginic antibody and usually referred to as the Type I. It occurs most commonly in atopic subjects and is characterized by changes in the airflow resistance in the conducting system of the lungs. This type of response has been described in the preceding discussion of diseases of the airways. The second and less common type of reaction affects the lung parenchyma, viz., the respiratory bronchioles and alveoli and does not appear to be related to atopy. Pepys coined the term extrinsic allergic alveolitis (hypersensitivity pneumonitides) to describe this response (1), which is also known as hypersensitivity pneumonitis.

CAUSATION

Although a large number of organic dusts have been identified as causes of extrinsic alveolitis, the pathophysiological effects are similar no matter which dust is responsible. The demonstration of specific precipitins in the serum of subjects affected by allergic alveolitis led Pepys to hypothesize that the basic mechanism in extrinsic allergic alveolitis is an Arthus or Type III reaction (1). Since most of the antigenic dusts

that produce extrinsic allergic alveolitis are organic, and since the serum of afflicted subjects often contain specific precipitins, there is an obvious resemblance to serum sickness. In the latter condition, the antigen is introduced into the body by injection, while in contrast, in extrinsic allergic alveolitis the antigen is inhaled.

Although not everybody who is repeatedly exposed to the antigen develops extrinsic allergic alveolitis, a small percentage does. Similarly, while there is good evidence that a substantial proportion of the subjects who are exposed to the antigen develop antibodies, the presence of antibodies alone is not necessarily an indication that the patient has or is likely to suffer from hypersensitivity allergic pneumonitis. Most subjects who develop allergic pneumonitis do so as a consequence of occupational exposure, but in some instances the person's avocation is more likely to be responsible.

The main differences between Type I and Type III pulmonary responses are shown in Table 1. It is important to add that since Pepys first called attention to the syndrome of extrinsic allergic alveolitis, its recognized causes have doubled or tripled (2).

CLINICAL FEATURES

Whatever the antigen, the clinical features of the condition are relatively similar. The acute form of the disease is more easily recognized and usually presents the symptoms of a sudden onset of fever, chills, shortness of breath, and a dry cough which appear between 4 to 8 hours following exposure. The patient may be severely distressed and on physical examination shows the presence of cyanosis, marked tachypnea, often of around 35 to 40 per minute, and diffusely scattered crepitations at both lung bases.

During the acute phase, pulmonary function tests show desaturated blood, reduced arterial PCO_2 , and a mild to moderate respiratory alkalosis. The lung volumes are greatly reduced, especially the vital capacity; however, there is no evidence of increased resistance to flow in the airways.

Measurements of the mechanical properties of the lungs indicate that the lungs are stiffer than normal and that their compliance is greatly reduced. The radiographic appearances are those of a diffuse acinous filling process predominantly affecting the mid and lower zones. The appearances in an air bronchogram are somewhat suggestive of pulmonary edema; however, there is no cardiac enlargement. Symptoms and signs gradually regress over a period of a week to ten days.

Besides the acute form of hypersensitivity pneumonitis, a more chronic form exists. This occurs with repeated lesser exposures, and although on the first two or three occasions there may be mild fever and chills, the continued lesser insults are not so obviously related to occupational exposure. In the chronic form which appears over a period of several months, the afflicted subject notices the onset of dyspnea, sometimes with occasional mild fever. This is usually accompanied by loss of weight, and general lethargy.

Table 1. Pulmonary response differences (2).

	Extrinsic allergic asthma (Type I)	Extrinsic allergic alveolitis (Type III)
Predisposing factors	Atopy	None known
Region affected	Conducting system of the lungs: bronchi to terminal bronchioles	Acini, respiratory bronchioles, and alveoli
Histology	Mucous plugging, bronchial edema, and eosinophilic infiltration	Granulomatous pneumonitis, occasionally undergoing organization and leading to interstitial fibrosis.
Onset of symptoms	Immediate	4 to 6 hours
Systemic reaction	None	Usual and accompanied by fever
Signs	Wheezes (rhonchi)	Crackles (crepitations)
Radiographic signs	Overdistension	Acinous filling pattern, often coexisting with some reticulonodulation in more chronic forms of the syndrome
Serological findings	Elevated IgE	Precipitins present (90% of cases)
Pulmonary impairment	Increased air flow resistance	Restrictive pattern
Eosinophilia	Common	Transient and uncommon
Skin tests	Immediate and urticarial	Edematous reaction appearing in 4 to 6 hours

Physical examination may reveal some cyanosis, clubbing may be present, and there may be diffuse scattered crepitations in both lower zones. The radiographic appearances are more suggestive of chronic interstitial fibrosis than of extrinsic allergic alveolitis. Pulmonary function tests in the subacute and chronic forms of the syndrome show restrictive disease with small lungs.

As the disease progresses, the lungs become smaller, the dyspnea worsens, and the end result resembles fibrosing alveolitis. In the established chronic case, the histological appearance cannot be distinguished from that seen in chronic interstitial fibrosis of unknown etiology. Both farmer's lung and pituitary snuff allergic alveolitis have been known to present a chronic interstitial fibrosis appearance.

PREVENTION

Elimination of personal exposure to the antigen can prevent the development or recurrence of the disease. This necessitates either environmental controls or personal protection. Environmental controls may include the elimination of conditions conducive to bacterial and fungal growth, process changes preventing the production of the antigen, or ventilation and particulate controls that eliminate contact of the antigen with the worker.

Personal protection can best be provided by the use of respirators to prevent inhalation of the antigen. The appropriate respirator should be selected on the basis of the characteristics of the dust or spores, the situation involved, and individual acceptance. When protection is inadequate for an individual with the hypersensitivity, removal of that individual from the offending environment is indicated.

PATHOLOGY

In the acute phase of the disease, the histological appearances of the lung show that the alveolo-capillary membrane is thickened, and that there is histiocytic, lymphocytic, and plasma cell infiltration. There may also be an edema-like fluid present in the alveoli. Numerous epithelioid tubercles may be seen but caseation necrosis is absent, and tubercle bacilli and fungi are not seen. The one exception to this is that occasionally in maple bark disease, *Cryptostroma corticale* spores may be seen in the lung parenchyma. Even so, maple bark disease is not a true fungal infection, but an allergy to the spores of this organism. The general appearance of extrinsic allergic alveolitis is that of a granulomatous interstitial pneumonia, with the granulomata bearing a resemblance to those seen in sarcoidosis.

Early in the disease, there is often an increase in the number of reticulin fibers but later on collagenous fibrosis predominates. A bronchiolitis affecting the respiratory bronchioles may also be present. Pathological changes found in chronic farmer's lung and other extrinsic allergic alveolitis are those of an interstitial fibrosis with collagenous thickening of the septa and lymphocytic infiltration. The fibrosis is often worse in the upper lobes and there may be frequent pigment laden macrophages present in the alveoli. Later on the intima of the pulmonary arteries is thickened and when this occurs pulmonary hypertension supervenes. In the terminal stages, cystic areas with honeycombing may be present in the lungs.

IMMUNOLOGY

The abrupt onset within three to four hours of exposure argues against this syndrome being an infective process. In addition, inhalation of aqueous extracts of mouldy hay will reproduce the clinical features of farmer's lung as will extracts of *Micropolyspora faeni*. The reaction develops several hours after the challenge and is associated with a

Table 2. Common clinical conditions (2).

Clinical condition	Source of offending agent	Precipitins against
Farmer's lung	Mouldy hay	<i>Micropolyspora faeni</i> <i>Thermoactinomyces vulgaris</i>
Baggassosis	Mouldy bagasse	<i>Thermoactinomyces vulgaris</i>
Mushroom worker's lung	Mushroom compost	<i>Micropolyspora faeni</i> <i>Thermoactinomyces vulgaris</i>
Suberosis	Cork dust	Cork dust
Maple bark disease	Maple bark	<i>Cryptostroma corticale</i>
Sequoiosis	Redwood sawdust	<i>Graphium Pullaria</i>
Papuan lung (New Guinea lung)	Mouldy thatch dust	Thatch of huts
Wood pulp worker's disease	Wood pulp	<i>Alternaria</i>
Malt worker's lung	Mouldy barley	<i>Aspergillus clavatus</i> <i>Aspergillus fumigatus</i>
Dog house disease	Mouldy straw	<i>Aspergillus versicolor</i>
Bird fancier's lung (Pigeon breeder's lung)	Pigeon, parrot and other bird droppings	Sera, protein, and droppings
Pituitary snuff taker's lung	Bovine and porcine pituitary snuff	Pituitary antigens
Wheat weevil disease	Wheat flour	<i>Sitophilus granarius</i>
Furrier's lung	Animal hairs	
Coffee worker's lung	Coffee bean	Coffee bean dust
Paprika splitter's lung	Paprika	
Lycoperdonosis	Puffball Lycoperdon Pyriform	

decline in ventilatory and diffusing capacities. In addition, there is a rise in the temperature of the patient which is usually accompanied by marked hyperventilation.

During the acute stage, precipitins are nearly always present in the serum of the affected subject, but with convalescence the titer often drops and may become negative if further exposure does not occur. Nevertheless, the presence of precipitins does not confirm the diagnosis of extrinsic allergic alveolitis; neither does their absence exclude it.

This syndrome is produced by a large number of different antigens, of which some of the more common ones are shown in Table 2. If a subject presents the clinical features of the syndrome, a detailed occupational history should be taken to see whether an antigen of organic nature in the patient's working environment might be responsible. It is also necessary, however, to stress that certain conditions develop as a result of the patient's avocation, e.g., pigeon fanciers' disease, pituitary snuff hypersensitivity pneumonitis. Therefore, if extrinsic allergic alveolitis is suspected, the patient's serum should be examined for precipitins against the offending antigens, and if present, the patient may be challenged with the aerosolized antigen and his ventilatory capacity or preferably his diffusing capacity assessed at intervals for four to eight hours following the challenge. If either falls, then the diagnosis of extrinsic allergic alveolitis can be made.

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PNEUMOCONIOSES

The word "pneumoconiosis" literally means dust in the lungs. Not all dusts that can be deposited in the lungs cause recognizable disease, so that the most widely accepted definition of pneumoconiosis is that of the International Labour Organization which states that "Pneumoconiosis is the accumulation of dust in the lungs and the tissue reaction to its presence. For the purpose of this definition, 'dust' is meant to be an aerosol composed of solid inanimate particles (1)."

DEPOSITION OF PARTICLES

Inhaled particles closely follow the movement of the air in which they are suspended as they are carried into the lungs during the inspiratory phase of respiration. They are, however, acted upon by certain forces which tend to promote their deposition within certain anatomic regions of the lung (2). The first of these, gravitational sedimentation, refers to the fact that the falling speed or terminal velocity of an airborne particle under the influence of gravity is proportional to its density and the square of its diameter. Thus, larger and more dense particles settle out more rapidly than smaller, less dense particles. This mechanism is responsible for most of the deposition of particles during breathing.

The second factor promoting deposition of particles in the lungs is inertial impaction and refers to the fact that a particle tends to maintain its original direction of travel despite a change in direction of the airstream in which it was suspended. This mechanism is largely responsible for the deposition of particles in the nose and at bifurcations of the lower airways.

The third factor promoting deposition of particles in the lungs is termed diffusion or, preferably, Brownian motion. All airborne particles are moving at random owing to their constant bombardment by gas molecules. In general, only the particles smaller than about 0.1 micron in diameter acquire sufficient Brownian motion to become deposited as a result of this mechanism alone.

Aerodynamic behavior refers to the mobility of particles regardless of their apparent size and shape. Thus, a relatively large, loosely aggregated clump of particles may behave aerodynamically in the same fashion as a much smaller dense particle. A fiber nearly 100 microns long but with a 3-micron diameter may behave in much the same fashion as a spherical particle about 3 microns in diameter as regards its ability to penetrate into the deeper regions of the lungs.

Other factors that appear to be important in determining the deposition of airborne particles are the pattern of breathing; namely, nose versus mouth breathing, and possibly individual variation in the filtration efficiency owing to differences in size and shape of the airways.

The International Commission on Radiological Protection has calculated the probabilities for the regional deposition of aerosols as a function of particle size based upon certain breathing patterns (3). In general, these curves show that the majority of particles larger than 15 microns will be deposited in the nose. However, as the particle size decreases to about 10 microns, an increasingly larger percentage will be deposited in the tracheobronchial tree; between about 5 microns and about 0.5 microns, the particles will be deposited in the alveoli and respiratory bronchioles. It is the deposition of particles of approximately 0.5 to 5 microns in the alveoli and respiratory bronchioles that gives rise to the group of diseases known as the pneumoconioses.

CLEARANCE OF PARTICLES

The conducting airways down to the terminal bronchioles are lined by a ciliated epithelium covered by a layer of mucus secreted by glands located in their walls and by goblet cells interspersed at intervals in the epithelium. The mucous layer is continuously propelled upward toward the mouth by the motion of the tiny hair-like cilia. Particles deposited in this layer are cleared usually within 24 hours to the oropharynx where they are swallowed along with the mucus. Wide variations in bronchial clearance are known to exist in man, but the reasons for these are not entirely clear (4).

Particles that penetrate to and are deposited in the alveolar areas of the lungs are cleared much more slowly than those deposited in the conducting airways. Evidence indicates that the particles are engulfed by alveolar macrophages and are somehow carried to the mucus escalator system to be cleared. The rate of clearance of an alveolar dust load probably depends to a large extent upon the availability of alveolar macrophages (5). The rate of alveolar clearance is also in part determined by the relative biological toxicity of the dust particles to the alveolar macrophages and, in part, by the total amount of dust already

present within the lungs (2). Nonetheless, both bronchial and alveolar clearance of dust particles are relatively efficient since abundant evidence exists that only a small fraction of the total dust load deposited in the lungs is retained in even the most advanced cases of pneumoconiosis.

PATHOLOGY

The different forms of pneumoconiosis are difficult to classify. One useful classification is that of Nagelschmidt in which four types of reaction appear to be distinguishable (6):

1. Hyaline-nodular fibrosis (classical silicosis)
2. Simple pneumoconiosis of coal miners
3. Mixed dust pneumoconiosis
4. Diffuse interstitial fibrosis

SILICOSIS

The characteristic lesion of silicosis is the silicotic nodule (7). In its simplest form the silicotic nodule consists of a central core of hyalinized reticulin fibers arranged in concentric layers which, towards the periphery, blend with coarser fibers of collagen to form a relatively distinct capsule. Early in the course of silicosis these nodules occur adjacent to, or in the walls of, the respiratory bronchioles. These nodules are thought to be formed by the death of macrophages laden with fine silica and the fibrosis resulting from the release of intracellular enzymes along with the ingested particles. The silica particles are ingested by new macrophages which are in turn killed, thereby releasing their potent intracellular enzymes to promote further fibrosis and, thus, the process becomes progressive (8).

The upper lobes and hilar lymph nodes are often more severely affected than the lung bases. In early stages, the nodules may remain isolated, but as the disease progresses the nodules crowd closer together until they appear to form a continuous mass of fibrous tissue. However, on close inspection, discreet nodules can usually be distinguished and what appeared to be a diffuse fibrosis is in reality many compressed nodules.

Silicosis not only favors the growth of tubercle bacilli, but may suppress the usual features of epitheloid cell proliferation, giant cell formation, and lymphocytic reaction to the extent that caseous necrosis in the center of a silicotic nodule may be the only indication of coexistent tuberculosis infection (9). Silicosis is also associated with pulmonary hypertension and cor pulmonale, probably partially as a consequence of damage by adjacent nodules to the walls of blood vessels which produces mechanical obstruction, and partially as a result of abnormal blood gas tensions leading to vasoconstriction.

COALWORKERS' PNEUMOCONIOSIS

The characteristic lesion in coalworkers' pneumoconiosis is the coal macule which consists of an accumulation of dust-laden macrophages

around respiratory bronchioles surrounded by a halo of dilated airspaces (10). In addition to the accumulations of coal in the macule, there is a slight increase in reticulin fibers and, to a lesser extent, collagen fibers. The presence of coal macules around the walls of the respiratory bronchioles may lead to atrophy or even to disappearance of the smooth muscle, this leading to a permanent dilatation of these small airways commonly called focal emphysema.

In about 1 to 2 percent of miners with simple dust accumulation, large, solid, black masses develop which represent accumulations of coal dust within macrophages and between reticulin and collagen fibers. These lesions are commonly formed in the upper lobes and differ from silicotic conglomerate masses in that the masses are not composed of discreet compressed nodules. The cause of the large lesions ("progressive massive fibrosis") in coal workers is not known. They are probably not due to coexisting tuberculous infection, but may represent an immunological reaction to the accumulated dust load. See Figure 5.

Caplan described the appearance of multiple rounded nodules in the lungs of coal miners with rheumatoid arthritis that subsequently proved to be necrobiotic nodules resembling those seen in rheumatoid arthritis (11). Microscopically, these lesions demonstrate a pale, necrotic center surrounded by granulomatous tissue having a typically "palisaded" appearance at the periphery of the nodule. Typical Caplan nodules have subsequently been reported in other occupations than coal mining, suggesting that they are not specifically related to coal dust exposure.

MIXED DUST PNEUMOCONIOSIS

In the mixed dust pneumoconioses the pathology depends to a large extent upon the relative proportion of free silica or quartz present in the airborne dust. Those with a quartz content of less than about 0.1 percent tend to develop small nodular areas in the lungs in almost direct proportion to the total amount of dust deposited, but little in the way of reticulin or collagen fibrosis, and very little emphysema. The pathological lesions more nearly resemble those found in coal miners.

On the other hand, dusts in which the quartz content ranges from about 2 percent to about 18 or 20 percent of the total dust tend to produce lesions that more nearly resemble those seen in classical silicosis.

Some examples of dusts that contain almost no free quartz are kaolin, talc, iron oxide associated with welding, coal, and coke used in making carbon electrodes.

DIFFUSE INTERSTITIAL FIBROSIS

There are a number of pneumoconioses that tend to produce diffuse interstitial fibrosis as their characteristic pathological lesion (6). Among these are berylliosis, aluminosis, Shaver's disease, and asbestosis. It appears likely that certain slowly dissolving constituents in the dusts give

rise to a peculiar disseminated interstitial fibrosis rather than to the focal or nodular types seen in coal miners or silicosis. As a general rule, the amount of dust found in the lungs in this type of pneumoconiosis is small and the fibrotic reaction that occurs is out of proportion to the amount of dust deposited.

RADIOGRAPHIC DIAGNOSIS

Unfortunately, the ability to diagnose the presence of pneumoconiosis during life is not as precise and clear-cut as the pathological responses described above. There are two general patterns of radiographic response recognizable. Both classical silicosis and the mixed dust pneumoconioses, including that seen in coal workers, tend to produce nodular opacities or a combination of reticular-nodular opacities on the chest roentgenogram. These are basically rounded shadows and are classified by type, profusion, and extent under the ILO U/C Classification of the pneumoconioses (12).

The pneumoconioses that produce the diffuse interstitial pathology are generally manifested as reticular and linear opacities on the chest roentgenograms. These likewise are generally classified by type, profusion, and extent as irregular opacities under the ILO U/C Classification of pneumoconiosis. Both rounded and irregular opacities may be present as the background upon which complicated pneumoconiosis or progressive massive fibrosis develops.

CLASSIFICATION

The ILO U/C Classification is intended to provide a simple reproducible means of systematically recording the radiographic changes associated with the inhalation of all types of mineral dusts. It is likely to be most useful in relating the radiographic features to indices of dust exposure and changes in lung function, particularly in epidemiological studies. It should also make possible comparison of data obtained in studies from other countries.

The system basically classifies the radiographic features by small (less than 1 cm) and large (greater than 1 cm) opacities; thus, simple pneumoconiosis is diagnosed when none of the opacities exceed 1 cm in diameter, and complicated pneumoconiosis is diagnosed when one or more of the opacities exceed 1 cm in diameter. Within the small opacity category, one recognizes the type, profusion, and extent of involvement of the lungs by the opacities.

The simple pneumoconiosis is further subdivided into small rounded opacities and small irregular opacities. The small rounded opacities are classified into types p, q, r, according to the approximate diameter of the predominant opacities. The p type includes rounded opacities up to about 1.5 mm in diameter; the q (m) type includes rounded opacities exceeding about 1.5 mm up to about 3 mm in diameter; the r (n) type includes rounded opacities exceeding about 3 mm and up to about 10 mm in diameter.

CATEGORY

Profusion refers to the number of small opacities per unit area. Thus, the lung fields are divided into three zones on each side, and the number of opacities within each zone is graded. Standard radiographs are available for comparison which divide the profusion into categories 0, 1, 2, and 3. Category 0 refers to the absence of opacities or the presence of less profuse opacities than in category 1; category 1 shows small rounded opacities present, but few in number, and the normal lung markings are usually visible; category 2 shows numerous small rounded opacities, and the normal lung markings are still visible; category 3 shows very numerous small rounded opacities, and the normal lung markings are partly or totally obscured.

Actually, there is a continuum of changes from normality to the most advanced category and, to recognize this, the British National Coal Board developed a 12-point scale (13). This scale permits subdivisions of profusion into finer grades and is useful in epidemiological studies where progression of pneumoconiosis is important. The radiograph is classified into one of the four categories in the usual way by comparison with the standard midcategory films. If, during the process, the category above or below was considered as a serious alternative, this is also recorded. Thus, if a category $\frac{1}{2}$ is recorded, it means that on comparison with standard radiographs the radiograph most nearly matched the category 1, but category 2 was seriously considered as an alternative.

The extent of pneumoconiosis is recorded by noting which of the lung zones are involved. Each lung is divided into three roughly equal zones by imaginary lines drawn at approximately one-third and two-thirds of the vertical distance between the apex of the lung and the dome of the diaphragm. Thus, each lung is divided into upper, middle, and lower zones for the purposes of recording the extent of pneumoconiosis.

IRREGULAR OPACITIES

Small irregular opacities are classified in much the same way as the small regular opacities, by type, profusion, and extent. Irregular opacities characteristically occur in asbestosis, but also occasionally in the other pneumoconioses. The variability, however, of these opacities in shape and width makes it virtually impossible to provide quantitative dimensions as is done in the rounded opacities; therefore, the types are divided on the basis of thickness. The s type refers to fine irregular, or linear, opacities; the t type refers to medium irregular opacities, and the u type refers to coarse (blotchy) irregular opacities. Standard radiographs of the three types of irregular opacities are available for comparison. Profusion of irregular opacities is graded in exactly the same way as is done in the rounded small opacities.

PLEURAL CHANGES

Certain pleural changes have recently become recognized as accompaniments to the parenchymal changes referred to above as part of

some pneumoconioses. Therefore, the ILO U/C Classification records pleural thickening by site (costophrenic angles, chest wall, diaphragm), width, and extent.

Pleural calcification is also classified by site and extent. When the cardiac outline and diaphragm are ill-defined, this is also recorded. A number of obligatory and optional symbols are also included in the classification for the benefit of a more complete description of the radiographic findings. The reader is referred to the complete ILO U/C Classification for details regarding these features which are beyond the scope of this chapter (12).

PHYSIOLOGICAL RESPONSES

The lungs have relatively few ways of responding to the dust burdens presented to them. Physiologically, two major patterns of response can be identified: an obstructive impairment and a restrictive impairment.

The obstructive pattern is characterized by a reduction in expiratory air flow, usually associated with either an increased airway resistance or a loss of lung recoil, or both. Increased airway resistance most commonly results from intrinsic narrowing of the airways owing to spasm of the smooth muscle in the walls (such as occurs in asthma), or to edema, inflammation, and mucus plugs (such as occurs in chronic bronchitis). These aspects have been more fully discussed in the chapter on airways mechanisms and responses.

OBSTRUCTIVE IMPAIRMENT

The obstructive pattern of physiological impairment in the pneumoconioses is more likely the result of localized or diffuse abnormalities in the lung recoil, owing to destructive changes in and around the small airways (less than 2 mm diameter) caused by the dust deposits. In simple pneumoconiosis these changes may be severe enough to cause alterations in the distribution of the inspired air and minor degrees of mismatching of ventilation and blood flow detectable only by using very sensitive techniques. These physiological impairments could prove disabling for heavy physical activities, but not for ordinary activities or at rest.

Spirometric tests of ventilatory capacity are usually within normal limits, or very nearly normal, in simple silicosis, simple coalworkers' pneumoconiosis, and the simple mixed dust pneumoconioses, unless asthma, chronic bronchitis, or emphysema, coexist. In the complicated form of the pneumoconioses (PMF), ventilatory capacity as measured by spirometry is often abnormal and consists of elements of both obstruction and restriction. Here, in addition to the loss of lung recoil owing to focal emphysema, some of the obstructive impairment may be attributable to distortion and kinking of airways by the large conglomerate masses. These large masses also generally interfere with gas exchange by reducing the surface area available for diffusion and the obliteration of the capillary bed.

Table 3. Agent, pathology, and impairment associated with pneumoconioses.

Agent	Type of Pathology	Type of Respiratory Impairment
1. Silica		
Simple	Nodular fibrosis	Restrictive, diffusion
Complicated	Conglomerate nodular fibrosis	Restrictive, obstructive, diffusion
2. Hematite	Nodular fibrosis	Restrictive, diffusion
3. Mixed dusts		
Iron and silica	Nodular fibrosis (Rarely conglomerate nodular fibrosis)	Restrictive, diffusion
4. Silicates	Nodular fibrosis (Rarely conglomerate nodular fibrosis)	Restrictive, obstructive
Talc		
Kaolin		
Bentonite		
Diatomite		
Tripoli		
Fuller's earth		
Mica		
Sillimanite		
Cement	Nonspecific bronchitis	Obstructive
5. Coal		
Simple	Peribronchiolar macules, focal emphysema	Obstructive (small airways)
Complicated	Conglomerate nodular fibrosis	Obstructive, restrictive, diffusion
6. Graphite	Peribronchiolar macules, focal emphysema	Obstructive (small airways)
7. Aluminum	Interstitial fibrosis	Restrictive, diffusion
8. Asbestos	Interstitial fibrosis	Restrictive, diffusion
9. Beryllium	Interstitial fibrosis (granulomata)	Restrictive, diffusion
10. Tungsten carbide	Interstitial fibrosis	Restrictive, diffusion
11. Barium	Simple dust accumulation	None known
12. Cerium	Simple dust accumulation	None known
13. Iron	Simple dust accumulation	None known
14. Tin	Simple dust accumulation	None known
15. Titanium	Simple dust accumulation	None known

The simple forms of classical silicosis, coal workers' pneumoconiosis, and the mixed dust pneumoconiosis, generally demonstrate mild obstructive impairment; whereas, the complicated forms (PMF) usually present mixtures of obstruction, restriction, and abnormalities of gas exchange.

RESTRICTIVE IMPAIRMENT

The restrictive pattern of physiological response is characterized by a reduction in lung volumes and ventilatory capacity, usually unaccompanied by an increased air flow resistance or hyperinflation. The restrictive pattern is also associated with an increased lung recoil, reduction in surface area for gas exchange and/or thickening of the air blood interface of the lungs.

The pneumoconioses that lead to diffuse interstitial fibrosis usually present the restrictive pattern of physiological impairment. In these, the earliest impairments are those involving gas exchange and diffusing capacity, and may be detectable only during exercise. In the later stages, gas exchange and diffusion abnormalities are detectable also at rest and are associated with a reduction in lung volumes, such as the vital capacity, total lung capacity, and the inspiratory reserve volume. Again, in cases where pneumoconiosis coexists with asthma or chronic bronchitis, this restrictive pattern may be associated with some element of obstructive impairment.

Asbestosis, berylliosis, aluminosis, and Shaver's disease are examples of pneumoconioses that are characteristically associated with the restrictive pattern of physiological impairment.

A summary of agent and type of pathology and respiratory impairment is given in Table 3.

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SECTION VI

What is a weed? A plant whose virtues have not been discovered.
— Ralph Waldo Emerson

PLANT AND WOOD HAZARDS

Tracy E. Barber, M.D., and E. Lee Husting, Ph.D.

Both local and systemic manifestations occur from occupational exposure to plants, plant products, and woods. Dermatitis is frequently observed; other effects include asthma, hay fever, irritations, toxic effects, and allergenic responses.

PLANTS AND PLANT PRODUCTS

Dermatitis due to plants of the genus *Rhus* is the most frequently seen allergic contact dermatitis in the United States. Plants in the genus *Rhus* include poison ivy, poison oak, and poison sumac. The contact dermatitis caused by these plants is identifiable by characteristic linear and bullous lesions. At least one of the species of *Rhus* is found in every part of the continental United States. Poison ivy occurs in every state but California where it is poison oak that has been the main cause of occupational skin disease. The oakleaf form of poison ivy and poison sumac are found mainly in the south and east, from Texas to New Jersey. Western poison oak occurs in Washington, Oregon, and California.

The genus *Rhus* belongs to the family Anacardiaceae which includes the cashew nut tree, the Japanese lac tree, the Indian marking nut tree, and the mango. A phenolic liquid is extracted from cashew nut shells, and this liquid, used to form resins for varnishes and brake shoe linings, is an irritant as well as a sensitizing agent. The fruit of the mango may cause contact dermatitis in *Rhus*-sensitive persons.

Dermatitis results from contact with the milky sap found in the roots, stems, leaves, and fruit of *Rhus* plants. In a dry atmosphere, the sap may retain its potency for months or perhaps years. The sap may be transmitted on soot particles when the plant is burned, or may be carried by animals, equipment, or apparel.

Allergic contact dermatitis may also be caused by the bastard feverfew (a common southeastern weed), English ivy, and castor bean plants. Allergic dermatitis may be caused by contact with certain flowers (such as primrose, chrysanthemum, poinsettia) and bulbs of hyacinth, narcissus, and tulips. The lipid fraction of ragweed pollen may cause eczematous dermatitis, while a water-soluble fraction may cause asthma or hay fever.

Contact dermatitis has occurred from handling fruits and vegetables, including carrots, asparagus, and some citrus fruits. Fruit and vegetable handlers may also suffer contact dermatitis due to insecticides and fungicides. Indirect effects of handling fruit and vegetables include chapping and moniliasis from exposure to moisture, photosensitization dermatitis from sunlight, and parasitism by mites.

Photosensitization is the delayed development of erythema, edema, vesicles, and bullae after contact with plant juices and exposure to sunlight. This accentuated localized sunburn is a phototoxic, rather than a

photoallergenic effect, and may result in either hyperpigmentation or depigmentation. Plants which cause photosensitization include fig, rue, lime, bergamot, parsnips, parsley, carrots, fennel, dill, and pink rot celery.

Hay fever, asthma, and urticaria frequently occur in castor bean processors, resulting from a potent allergen found in the dried pomace remaining after castor oil extraction. Castor bean workers, dock workers handling the pomace, or farmers using the pomace for fertilizer may be affected.

Historically, paprika sorter's disease was frequent in women splitting paprika fruit who inhaled spores and mycelia of a mold growing in the fruit. This exposure has been eliminated since the entire fruits are now ground mechanically.

Exposure to grain dusts may result in coughing, wheezing, breathlessness, dermatitis, and grain fever. The incidence of these symptoms is higher in individuals with a history of past allergy, suggesting that allergy may be partly responsible for the response to grain dusts.

Tobacco cropper's or green-tobacco sickness, characterized by weakness, nausea, and vomiting, has been observed in persons pulling tobacco leaves from the plants during cropping. It is believed that a noxious material in green tobacco gum, most likely nicotine, is absorbed through the skin.

POTENTIAL OCCUPATIONAL EXPOSURES

Agricultural workers	Gardeners
Botanists	Grain elevator workers
Bulb handlers, plant	Highway workers
Camp workers	Hop pickers
Canners	Horticulturists
Castor bean workers	Loggers
Construction workers	Pipeline workers
Dock workers	Road builders
Field laborers	Surveyors
Flower cutters	Telephone linemen
Flower packers	Tobacco croppers
Foresters	Utility workers
Fruit pickers	Vegetable harvesters
Fruit processors	Vegetable processors

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WOODS

Woods, wood dusts, and substances from woods may be toxic, irritant, allergenic, or carcinogenic.

Toxic woods, such as East Indian satinwood, South African boxwood, and ipe, contain substances which cause systemic signs and symptoms when absorbed, inhaled, or ingested. Wood toxins are often alkaloids. Effects may include headache, anorexia, nausea, vomiting, bradycardia, dyspnea, or somnolence.

Irritant woods cause injury to mucous membranes upon contact, and severe irritants may affect intact skin, causing dermatitis. Examples of irritant woods are mansonia, dahoma, and cocobolo.

Allergenic woods such as certain members of the birch, pine, dogwood, beech, mahogany, mulberry, and myrtle families may cause allergic manifestations including asthma and contact dermatitis in sensitized individuals.

It is believed that the inhalation of fine dusts from wood, especially hard wood dust, causes nasal cancer. Many woodworkers in the furniture industry develop squamous metaplasia in the nasal mucous membrane.

Furniture workers frequently exhibit an allergic response to western red cedar. The response occurs after contact with the sawdust of this wood, and symptoms are intensified by contact with the wood. Symptoms include asthma, rhinitis, urticaria, dermatitis, and conjunctivitis. Asthma and rhinitis are frequent in carpenters, while conjunctivitis occurs more often in sawmill workers.

POTENTIAL OCCUPATIONAL EXPOSURES

Cabinet makers	Musicians
Carpenters	Sawmill workers
Furniture makers	Violin makers
Lumbermen	Wood workers

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